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THE IMPACT OF OMEGA-3 FATTY ACIDS, VITAMINS, MINERALS, N-ACETYLCYSTEINE, AND GINKGO BILOBA ON THE COURSE AND TREATMENT OF SCHIZOPHRENIA: A REVIEW OF RESEARCH AND THERAPEUTIC PERSPECTIVES

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ABSTRACT
Schizophrenia is a mental disorder with a complex etiology involving both genetic and environmental factors. Despite advancements in antipsychotic treatments, many patients continue to experience chronic symptoms and medication side effects. As a result, there is growing interest in dietary supplements and bioactive substances to complement traditional therapies and improve patient quality of life. Omega-3 fatty acids, vitamins, minerals, N-acetylcysteine, and ginkgo biloba have shown potential in supporting schizophrenia treatment. These substances may help regulate neuroinflammation, neurotransmission disturbances, oxidative stress, and improve cognitive function. This review examines the scientific evidence on their effects and explores the mechanisms behind their potential benefits in schizophrenia management.

Keywords: "omega-3 fatty acids", "vitamins", "minerals", "N-acetylcysteine", and "Ginkgo Biloba", "gut-brain axis", "antipsychotic medications"

Introduction

Schizophrenia is one of the most severe mental disorders, characterized by disturbances in perception, thinking, emotions, and behavior. The etiology of this disorder is complex, involving both genetic and environmental factors. Despite advancements in the treatment of schizophrenia, primarily based on antipsychotic medications, many patients continue to struggle with chronic symptoms and side effects of pharmacotherapy. As a result, increasing attention has been given in recent years to the role of dietary supplementation and bioactive substances that may support traditional treatments for schizophrenia and improve the quality of life of patients.

In particular, omega-3 fatty acids, vitamins, minerals, N-acetylcysteine, and ginkgo biloba are promising areas of research concerning their impact on the health of patients with schizophrenia. Omega-3 fatty acids, especially

DHA and EPA, have well-documented neuroprotective and anti-inflammatory properties, which may play a key role in modulating neuroinflammatory processes and neurotransmission disturbances typical of schizophrenia. Vitamins and minerals, such as vitamin D, B12, folate, and magnesium, significantly affect the health of the nervous system, and their deficiencies may exacerbate psychotic symptoms. N-acetylcysteine, with its antioxidant and detoxifying properties, shows promising effects in treating neuroinflammatory disorders and oxidative stress, which are present in the pathophysiology of schizophrenia. Additionally, ginkgo biloba, with its properties of improving cerebral circulation and antioxidant effects, may contribute to the improvement of cognitive functions and reduction of psychotic symptoms in patients.
The aim of this publication is to review the available scientific evidence regarding the impact of these substances on the health of patients with schizophrenia. Through an analysis of clinical and preclinical studies, this work seeks to assess the potential benefits of supplementation and identify the mechanisms that may explain their supporting role in the traditional treatment of schizophrenia.

The purpose of research
The aim of this publication is to analyze the available research on the impact of omega-3 fatty acids, vitamins, minerals, N-acetylcysteine, and Ginkgo Biloba on the mental health of individuals with schizophrenia, with particular focus on biological mechanisms such as the reduction of chronic inflammation, improvement of neurotransmitter balance, and gut microbiota. Additionally, the review will explore how these substances may interact with antipsychotic medications and potentially enhance their therapeutic effects. By understanding these mechanisms, the research aims to identify new avenues for adjunctive treatments that could improve both the clinical outcomes and overall well-being of patients with schizophrenia.

Research materials and methods
To optimize our review, on 20.02.2025 we used PubMed scraping script run in Python environment to create a database of all publications including keywords: “omega-3 fatty acids”, “vitamins”, “minerals”, “N-acetylcysteine”, and “Ginkgo Biloba”, “gut-brain axis”, “antipsychotic medications”. We excluded publications in languages other than English, we have not included year criteria.

Omega-3 fatty acids
Omega-3 fatty acids, particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), may play an important role in the function of the gut-brain axis, making them a promising component for supporting the treatment of neurological and psychiatric disorders. With their potential anti-inflammatory and antioxidant properties, they may influence the reduction of inflammation in the brain and gut microbiome, supporting neurotransmitter production, blood-brain barrier function, and neurological health (1) .

In the case of schizophrenia, omega-3s have shown potential in alleviating negative symptoms, such as social withdrawal, and may support traditional therapies in the early stages of the disease. At the same time, a diet rich in omega-3s may promote a healthy microbiome, which in turn may benefit neurotransmission processes and protect the brain from damage (2) .
Findings support the possible positive effects of omega-3 fatty acids on schizophrenia (3) (4) . Contemporary neuroscience research indicates that polyunsaturated fatty acids (PUFAs), especially omega-3 fatty acids such as eicosapentaenoic (EPA) and docosahexaenoic (DHA), play an important role in the pathophysiology of schizophrenia (5) . Of key importance is their effect on the integrity of neuronal cell membranes, which regulates receptor activity, synaptic conduction, and the dynamics of neurotransmitters, namely dopamine, glutamate, and serotonin. PUFA deficiency may interfere with these processes, promoting psychotic symptoms (6) .

In a study by Yao et al., the levels of IL-6 and IL-10 in cerebrospinal fluid and PUFAs in red blood cell membranes were measured in patients with schizophrenia. A significant inverse correlation was found between the levels of IL-6 and PUFAs in the membranes, both in haloperidol-treated and non-treated patients. This phenomenon mainly involved n-6 fatty acids, i.e. linoleic acid and arachidonic acid, and to a lesser extent n-3, i.e. eicosapentaenoic acid and docosahexaenoic acid. The results suggest that reduced levels of PUFAs in membranes may be associated with immune disorders in schizophrenia (7) .
According to the study by Xia et al., omega-3 fatty acids, particularly EPA and DHA, have shown the ability to modulate neuroinflammatory pathways by affecting the production of pro-inflammatory cytokines, such as IL-6, TNF-α, interleukin-1 beta (IL-1β), interleukin-8 (IL-8), and IFN-γ, as well as other inflammatory mediators such

as monocyte chemoattractant protein-1 (MCP-1) and prostaglandin E2 (PGE2), which are often elevated in patients with schizophrenia (8) . EPA and DHA act as substrates for the synthesis of anti-inflammatory lipid mediators such as class 3 prostaglandins and resolvins, which promote the inhibition of inflammatory processes in the CNS (9) .

A study by Xu et al. analyzed the effects of omega-3 fatty acids on metabolic disorders in patients with schizophrenia and metabolic syndrome (MetS). After 12 weeks of omega-3 supplementation, patients treated with olanzapine showed an improvement in triglyceride metabolism and a decrease in TNF-α levels, which was positively correlated with a decrease in TG levels (10) .

Similarly, Tang et al. investigated the effects of omega-3 fatty acids on cognitive function and BDNF levels in patients with schizophrenia and MetS. Omega-3 fatty acids increased BDNF levels and decreased inflammatory parameters, such as CRP, IL-6, and TNF-alpha, after 12 weeks of supplementation. The changes in the BDNF levels seemed to be negatively correlated with the changes in the TNF-alpha levels, whereas no significant correlation was found with the changes in the CRP and IL-6 levels (11) .

Dysfunction of the intestinal barrier, which is often observed in schizophrenia, may further exacerbate inflammation by increasing the translocation of lipopolysaccharides (LPS) into the systemic circulation [82]. Omega-3 PUFAs reduce oxidative stress by reducing the production of reactive oxygen species (ROS) and the activity of pro-inflammatory pathways such as MAPK (mitogen-activated protein kinases), NF-κB (nuclear factor kappa B), and AP-1 (activator protein-1) (12) . In particular, inhibiting MAPK-related kinases such as ERK (extracellular signal-related kinase) and JNK (c-Jun N-terminal kinase) helps to reduce pro-inflammatory responses in intestinal cells (13) .

The effect of supplementation with EPA and vitamins C and E in combination with antipsychotic treatment in patients with schizophrenia was investigated in a study by Bentsen et al. The study showed that in patients with low PUFA levels, supplementation with EPA and vitamins alone worsened psychotic symptoms, especially persecutory delusions, and increased the risk of treatment discontinuation. The worsening effect was neutralized when EPA was combined with vitamins E and C. The mechanism of action of the supplements was found to be related to oxidative stress. Oxidative stress exacerbates schizophrenia symptoms, especially in patients with low PUFA levels. High doses of EPA and vitamins C and E can have a psychotropic effect by disrupting the reduction-oxidation balance, leading to an increase in oxidative stress (14) .

Omega-3 fatty acids also help improve neuroplasticity and promote neuronal regenerative mechanisms by activating BDNF pathways (15) .

The effect of supplementation with omega-3 fatty acids (EPA and DHA) on the symptoms of first-episode schizophrenia was investigated in a study by Pawełczyk et al. It was shown that supplementation with omega-3 fatty acids (EPA and DHA) as an adjunct to antipsychotic treatment can contribute to a significant improvement in the symptoms of schizophrenia in patients in their first episode of the illness. In addition, this supplementation had a positive effect on the reduction of depressive symptoms and the improvement of patients' overall functioning (16) .

The effect of omega-3 supplementation on psychotic symptoms in patients with early-onset psychosis treated with risperidone was analyzed in a study by Robinson et al. The participants in the study were also treated with lorazepam for severe symptoms of anxiety. The results showed that the patients who took omega-3 fatty acids showed a significant improvement in symptoms of depression and anxiety, especially in the group that did not take lorazepam. Compared to the placebo group, significant differences in the BPRS scale scores were observed in these patients, with a significant improvement in depressive and anxiety symptoms. The study suggests that, particularly in the context of depressive and anxiety symptoms, omega-3 supplementation may be an effective adjunct to treatment in the early stages of psychosis (17) .

Bošković et al. conducted a study to assess the effect of supplementing vitamin E and EPUFAs in patients with schizophrenia treated with haloperidol. The study showed that the EPUFA group had a higher concentration of reduced glutathione, and the vitamin E group had a lower concentration of oxidized glutathione compared to the placebo group. The patients who took vitamin E had less motor slowing, but no differences were observed in terms of extrapyramidal symptoms (18) .

Selected vitamins and minerals

Vitamins and micronutrients such as folic acid, vitamin B12, and selenium may play an important role in alleviating the symptoms of schizophrenia. Folic acid and vitamin B12 are key components of the methylation pathway, which affects the production and metabolism of neurotransmitters such as dopamine and serotonin. Deficiencies in these vitamins can lead to hyperhomocysteinemia. Supplementing folic acid and vitamin B12 deficiencies can help improve the function of the methylation axis. Selenium, on the other hand, is a trace

element with strong antioxidant properties and is a key component of the enzyme glutathione peroxidase, which protects nerve cells from oxidative stress.

Folic acid deficiency may impact neurotransmitter processes, which in turn may exacerbate schizophrenia symptoms. According to Sakuma et al., folic acid supplementation has been shown to be beneficial in treating schizophrenia by improving response to antipsychotic medication (19) .

Selenium deficiency, which affects antioxidant enzymes such as glutathione peroxidase (GPx), can lead to increased oxidative stress and neuronal damage. This is associated with the risk of developing schizophrenia (20) . Studies have shown that reduced serum selenium levels are correlated with increased symptoms of schizophrenia, particularly negative symptoms such as apathy and cognitive impairment, as well as damage to brain structures (21) .

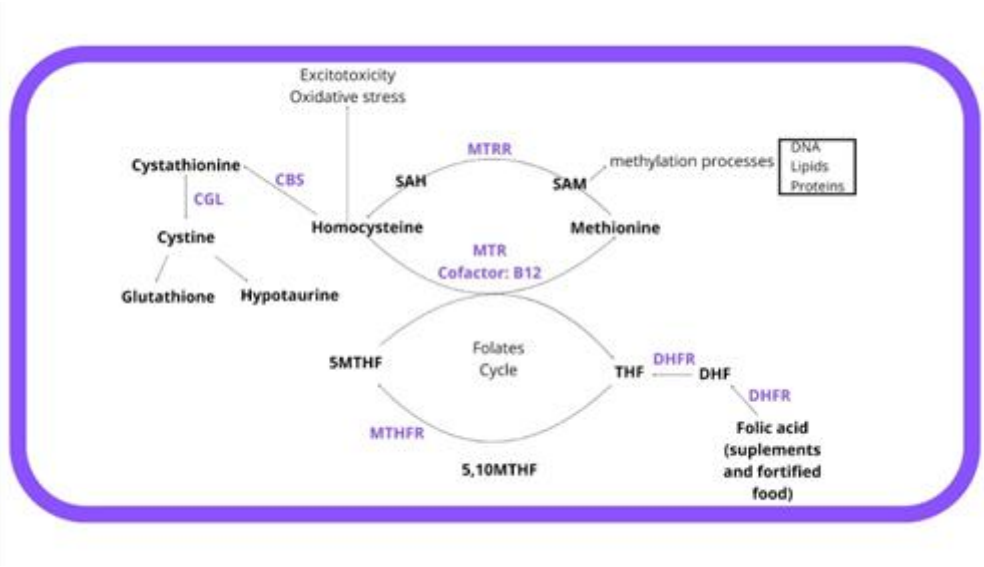
Selenium deficiency, by affecting the activity of enzymes like tyrosinase and the dopamine transporter, may interfere with dopamine metabolism, which is central to the pathophysiology of schizophrenia (22) . This may be associated with cognitive decline and increased positive symptoms of schizophrenia, including hallucinations and delusions. A neuroprotective role for selenium in this disease has been suggested, as high dietary selenium levels may modulate dopamine metabolism and reduce MAO activity (23) .

To investigate whether folic acid and vitamin B12 supplementation can reduce the negative symptoms of schizophrenia and how treatment response is influenced by genetic variants related to folate metabolism, Roffman et al. conducted a study. The trial involved 140 patients with stable chronic schizophrenia who were given 2 mg of folic acid and 400 µg of vitamin B12 daily or a placebo for 16 weeks. The results showed that supplementation improved negative symptoms compared to placebo, particularly in patients homozygous for the 484T allele of the FOLH1 gene. There were no significant changes in positive symptoms or in general symptoms (24) .

According to Zhilyaeva et al., vitamin B9 (folic acid) and B12 deficiencies in patients with schizophrenia can lead to elevated homocysteine levels, which increase oxidative stress and cell damage. Folate supplementation, especially in patients with relevant genetic variants, can improve the negative symptoms of schizophrenia and promote homocysteine metabolism. This suggests a potential role for this vitamin in the treatment of the disorder (25) .

The MTHFR 677C > T genotype causes reduced methylenetetrahydrofolate reductase enzyme activity, which interferes with folic acid metabolism and leads to elevated homocysteine levels. These changes may impact the development of schizophrenia, particularly the negative symptoms, by disrupting neurotransmitter function, which may exacerbate schizophrenia symptoms (26) .

Scheme 3. Homocysteine and folate pathways.



THF (tetrahydrofolate) is the active form of folate, playing a crucial role in carrying monocarbons for various metabolic reactions. It is produced through the reduction of dietary or supplemental folic acid by the enzyme dihydrofolate reductase (DHFR). THF can then be converted into 5,10-methylene THF. The enzyme MTHFR (methylenetetrahydrofolate reductase) converts 5,10-methylene THF to 5-methyl THF. Mutations in the MTHFR

gene can affect folate metabolism.
5-Methyl THF (5MTHF) is essential for the remethylation of homocysteine to methionine. This process requires methionine synthase (MTR), which uses vitamin B12 as a cofactor. Methionine, in turn, is a precursor of SAM (S-adenosylmethionine), the body's primary methyl donor. After donating a methyl group, SAM is converted to S-adenosylhomocysteine (SAH), which is then hydrolyzed to homocysteine.
The homocysteine remethylation cycle is a key metabolic process in the metabolism of folic acid and vitamin B12. It plays an important role in methionine synthesis and the regulation of blood homocysteine levels. When homocysteine levels are elevated due to deficiencies in folic acid and vitamin B12, oxidative stress and cell damage increase.
Some homocysteine does not re-enter the remethylation cycle but instead enters the transsulfuration pathway. In this pathway, the enzyme CBS (cystathionine β-synthase) converts homocysteine into cystathionine. Cystathionine is then broken down into cysteine, which is used in glutathione synthesis.

Shortcuts: THF – Tetrahydrofolate, DHF – Dihydrofolate, DHFR – Dihydrofolate reductase, 5,10MTHF – 5,10-methylene-tetrahydrofolate, 5MTHF – 5-methyl-tetrahydrofolate, MTHFR – Methylenetetrahydrofolate reductase, MTR – Methionine synthase, MTRR – Methionine synthase reductase, SAM – S-adenosylmethionine, SAH – S-adenosylhomocysteine, CBS – Cystathionine ß-synthase, CGL – Cystathionine ß-lyase

The study by Hill et al. explored the impact of folic acid supplementation on the negative symptoms of schizophrenia, considering the MTHFR 677C > T genotype. While supplementation did not significantly affect symptoms in the entire group, patients with the T allele showed greater improvements in negative symptoms with higher concentrations of folic acid. This suggests that the MTHFR genotype may moderate the response to folic acid supplementation (27) .
Vitamin D also plays a role in the treatment of schizophrenia. Low vitamin D levels are believed to increase the risk of developing schizophrenia and may contribute to the severity of symptoms (28) . Vitamin D influences brain function, and supplementation could potentially improve cognitive function and reduce depressive symptoms in patients (29) .
A study by Kalejanhi et al. examined the effects of vitamin D supplementation on metabolic factors linked to insulin resistance and chronic vitamin D deficiency symptoms. In the vitamin D-supplemented group, there was a significant reduction in waist circumference, positive and negative symptoms, and protein kinase GSK-3β levels. The placebo group, on the other hand, saw an increase in fasting insulin levels and HOMA-IR (30) .

In a meta-analysis by Valipour et al., the mean 25-hydroxyvitamin D level in schizophrenia patients was found to be significantly lower by 5.91 ng/mL compared to controls. Additionally, 65.3% of schizophrenia patients were vitamin D deficient, with those deficient having a 2.16 times higher risk of developing schizophrenia (31) .
A study by McGrath et al. found that vitamin D supplementation during the first year of life was associated with a reduced risk of developing schizophrenia in men, particularly at doses exceeding 2,000 IU. However, no such association was observed in women. These findings suggest that preventing vitamin D deficiency in early childhood may reduce the risk of schizophrenia, especially in males (32) .
Li et al. conducted a study analyzing trace element levels (including selenium, manganese, chromium, calcium, copper, arsenic, potassium, etc.) in schizophrenia patients compared to healthy individuals. The results showed that levels of selenium, copper, manganese, and calcium were lower in schizophrenia patients, while arsenic, potassium, boron, magnesium, and chromium were higher than in controls. Selenium supplementation was found to reduce arsenic levels and increase selenium and copper levels, leading to improvements in symptoms such as appetite and memory. The study suggests that selenium, manganese, chromium, calcium, and copper may have beneficial effects in schizophrenia, while arsenic and potassium may be harmful (33) .
The impact of probiotic and selenium supplementation on clinical and metabolic symptoms in patients with chronic schizophrenia was explored in a study by Jamilian et al. This study found that probiotic/selenium supplementation improved PANSS (Positive and Negative Syndrome Scale) scores, antioxidant capacity, glutathione levels, and reduced CRP, fasting glucose, insulin, insulin resistance, and insulin sensitivity (34) .

The Nechifor study analyzed the levels of magnesium, calcium, copper, and zinc in the plasma and magnesium in the erythrocytes of patients with paranoid schizophrenia, alongside the effects of treatment with haloperidol and risperidone. Erythrocyte magnesium levels were found to be reduced in 56 patients, while plasma magnesium levels remained unchanged. The study suggests that the Cu(2+)/Mg(2+) ratio in red blood cells and the Cu(2+)/Zn(2+) ratio in plasma may serve as important markers for acute paranoid schizophrenia (35) .

Selected Nootropic supplements- N-acetylcysteine and Ginco Biloba

Nootropics play an important role in human health research, especially in the context of protection against oxidative stress and neurodegenerative processes. N-acetylcysteine (NAC) and Ginkgo biloba are two well-studied compounds that have attracted the attention of researchers because of their potential therapeutic properties.NAC is a derivative of cysteine and a potent glutathione precursor. Glutathione dysfunction is a significant pathophysiological mechanism in schizophrenia, and studies have shown that NAC may have a beneficial effect in regulating glutathione (GSH) levels, potentially leading to improvements in schizophrenia symptoms (36) .

Ginkgo biloba is a plant that has shown potential as an adjunctive therapy in the treatment of schizophrenia symptoms. Due to its terpene content, including ginkgolide B, it acts as a neuroprotectant, an antioxidant, a free radical scavenger, a stabilizer of the cell membrane, and an inhibitor of the platelet-activating factor (PAF) (37, 38) . The plant also affects vasodilation by inhibiting cAMP phosphodiesterase, leading to endothelial relaxation (39) . Additionally, Ginkgo biloba protects muscarinic and adrenergic receptors from the loss of function associated with aging and stimulates the reuptake of choline in the hippocampus (40) . Studies have also shown that Ginkgo biloba can inhibit the deposition of beta-amyloid, which is significant in the context of neurodegenerative diseases (41).

Changes in glutathione levels in the prefrontal cortex may s in schizophrenia. According to a study by Yang et al., increased glutathione levels following 8 weeks of N-acetylcysteine supplementation in the medial prefrontal cortex may indicate a potential link to improvements in symptoms and cognitive function in the long term (42) .

A study by Pyatoykin on NAC in patients with paranoid schizophrenia showed that NAC supplementation (2000 mg per day for 60 days) significantly improved negative symptoms, general psychopathology, and cognitive function—particularly working memory—compared with a placebo. NAC treatment was also associated with increased GSH levels, although no significant differences in GSH levels were observed between the groups (43) .

The effect of NAC supplementation on symptoms and cognitive function in patients with early psychosis was investigated by Conus et al. After 6 months of supplementation (2700 mg/day), NAC had no effect on negative or positive symptoms. However, it improved cognitive function, especially information processing. Supplementation increased glutathione levels in the brain by 23% and in peripheral blood cells by 19%. Changes in positive symptoms were associated with redox reaction changes in a subgroup of patients with high glutathione peroxidase activity (GPxBC) (44) .

NAC has the ability to cross the BBB, where it increases the concentration of GSH, which is particularly important for neuroprotection in schizophrenia (45).

In a study by Mullier et al., the effects of NAC supplementation on functional brain connectivity within the cingulate cortex were investigated in schizophrenic patients with early psychosis. The results showed that NAC supplementation for 6 months led to an increase in functional connectivity along the cingulum, especially between the caudal anterior part and the isthmus of the cingulate cortex. An increase in the centrality of these areas in the functional brain network may partly explain these changes. This study suggests that NAC supplementation could improve functional brain connectivity by increasing glutathione levels in the brain, which may have beneficial effects in patients with early psychosis (46) .

In contrast, a study by Neill et al. evaluating the efficacy of NAC as adjunctive treatment in clozapine-resistant schizophrenia found that supplementation with 2g/day of NAC for one year had no significant effect on negative symptoms, cognitive function, or quality of life in this group (47) .

In a meta-analysis of six trials, Ginkgo biloba showed moderate improvement in both general and negative symptoms of schizophrenia when added to antipsychotic medication (48) .

The efficacy of Ginkgo biloba extract (EGb) in combination with haloperidol in patients with chronic treatment-resistant schizophrenia was evaluated in a study by Zhang et al. After 12 weeks of treatment, patients taking EGb plus haloperidol showed a significant reduction in both positive symptoms. Furthermore, 57.1% of patients taking EGb achieved a positive treatment response, a higher percentage than in the placebo group, where only 37.7% of patients had a response. There was also a significant reduction in treatment-related neurological symptoms in the EGb group (49) .

The use of EGb as an adjunct to clozapine in the treatment of treatment-resistant schizophrenia was investigated in a similar study by Doruk et al. The results showed that EGb was effective in reducing the negative symptoms of schizophrenia, achieving a greater mean reduction than placebo. Positive symptoms and the patients' general condition were not affected by EGb (50) .

According to Montes et al., Ginkgo biloba may be a valuable adjunct in the treatment of schizophrenia, especially when traditional antipsychotics do not provide sufficient improvement, due to its neuroprotective properties and beneficial effects on negative symptoms (51).
The efficacy of Ginkgo biloba extract (EGb-761) in the treatment of tardive dyskinesia (TD) in patients with schizophrenia was investigated in a study by Zhang et al. The results showed that EGb-761 significantly reduced TD symptoms compared to placebo. However, schizophrenia symptoms and cognitive test scores did not differ between the groups (52) .

Table 2. Potential effects of dietary interventions on brain function and psychiatric symptoms

Dietary Intervention	Mechanism of Action	Clinical Effects
Probiotics and Prebiotics	- Modulation of the gut-brain axis - Increased production of SCFAs (e.g., butyrate) - Reduction of inflammation and oxidative stress	- Improved cognitive function - Reduced CRP - Improved metabolic markers (glucose, HOMA-IR, lipids)
Omega-3 (EPA, DHA)	- Impact on neuronal membrane integrity - Reduction of inflammation (TNF-α, IL-6) - Decreased oxidative stress - Support for neuroplasticity through activation of the BDNF pathway	- Lower triglycerides and inflammatory markers - Improved cognitive function -reduction of psychotic, depressive and anxiety symptoms - Improved BPRS scores and reduced symptoms of early psychosis
Vitamin B (B9, B12)	- Reduction of homocysteine (enhanced neurotransmitter function) - Regulation of methylation pathways (folate metabolism) - Reduction of oxidative stress	- Reduced negative symptoms in patients with the MTHFR 677C>T variant - Improved response to antipsychotic treatment - Improved cognitive function
Vitamin D	- Regulation of pro-inflammatory cytokines (TNF-α, IL-6) - Reduction of oxidative stress	- Reduced positive and negative symptoms - Improved cognitive function and depressive symptoms
Selenium	- Cofactor in antioxidant enzymes (Gpx) - Modulation of dopamine metabolism and MAO activity - Reduction of oxidative stress	- Improved cognitive function - Reduced CRP levels

N-acetylcysteine (NAC)	- Precursor of glutathione (GSH), reducing oxidative stress - Improved functional connectivity of the brain - Neuroprotection through increased GSH in the brain	- Improved cognitive function and working memory - Reduced negative symptoms
Ginkgo biloba	- Neuroprotection via antioxidant properties - Stabilization of neuronal membranes - Modulation of muscarinic and adrenergic receptors	- Reduced negative symptoms of schizophrenia - Improved cognitive function - Reduced neurological side effects associated with treatment

Shortcuts: NAC - N-acetylcysteine, EPA - Eicosapentaenoic acid, DHA - Docosahexaenoic acid, TNF-α - Tumor necrosis factor alpha, IL-6 - Interleukin-6, BDNF - Brain-derived neurotrophic factor, CRP - C-reactive protein, HOMA-IR - Homeostasis model assessment of insulin resistance, GSH - Glutathione, Gpx - Glutathione peroxidase, MAO - Monoamine oxidase, BPRS - Brief Psychiatric Rating Scale.

Conclusions

The findings of current research indicate the promising potential of omega-3 fatty acid supplementation, NAC, Ginkgo biloba, as well as vitamins and minerals in the treatment of schizophrenia. Omega-3 fatty acids, especially EPA and DHA, may aid treatment by improving neuronal membrane integrity, regulating pro-inflammatory cytokine levels, and reducing oxidative stress. The use of NAC and Ginkgo biloba may, in turn, improve cognitive function and alleviate negative symptoms of schizophrenia, particularly in cases resistant to traditional antipsychotic medications. At the same time, deficiencies in vitamins and minerals, such as folic acid, vitamin B12, vitamin D, and selenium, can significantly impact the development and course of schizophrenia. Supplementing these substances may support treatment by reducing negative symptoms and improving cognitive function. Despite these promising results, further research is needed to precisely determine optimal dosages and the long-term effectiveness and safety of these therapies. Integrating these therapeutic strategies into schizophrenia treatment may contribute to a more comprehensive approach, improving patients' quality of life and their response to treatment.

Disclosure

Author's contribution

Conceptualization: Patrycja Niczyporuk, Rafał Marecki; Methodology: Patrycja Niczyporuk; Software: Izabela Zajkowska; Wiktor Królikiewicz; Check: Izabela Zajkowska; Formal analysis: Patrycja Niczyporukt; Investigation: Patrycja Niczyporuk; Resources: Patrycja Niczyporuk, Kamila Iłendo; Data curation: Izabela Zajkowska, Kamila Iłendo; Writing-rough preparation: Aleksandra Giba.; Writing-review and editing: Aleksandra Kicman, Rafał Marecki; Visualization: Aleksandra Giba; Supervision: Aleksandra Kicman, Rafał Marecki; Project administration: Aleksandra Kicman, Rafał Marecki

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